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# TWO CASES OF TORSION OF THE CECUM AND ILEUM IN RATS[1,2,3]

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SUMMARY • *Two male Caw:CFE(SD)SPF rats, 10 weeks old, died unexpectedly 24 hr apart while on nutrition studies. Necropsy revealed an enlarged, gas-distended, hemorrhagic cecum, with a 630° rotation at the ileo-cecal-colic junction, complicated by a secondary wrapping of the ileum around the colon.*

Torsion of the cecum is most commonly a medical problem of the larger domestic species (1–4). In the horse the torsion is said to occur as a result of rolling and/or an acute attack of impaction or spasmodic colic (3). In lambs, pigs, and calves, torsion of the cecum and rotation of the colon about its mesentery have been described (3). Laboratory workers are familiar with cecal torsion in the gnotobiotic rat. Several reports deal with the influence of diet and intestinal microflora on cecal size in the gnotobiotic rat (5–11). This report discusses the necropsy findings of 2 cases of rotation of the cecum with secondary involvement of the small intestine in the non-gnotobiotic rat. Several experimental attempts to induce the condition are also discussed.

## CASE REPORT

One hundred weanling rats, Caw: CFE (SD) SPF, were received for use in the bioassay of iron availability by the method of Pla and Fritz (12). The rats were examined on arrival and found healthy. The nutrition study involved producing an iron deficiency anemia by feeding an iron-deficient diet for 4 weeks and subsequently measuring the availability of iron from various sources in experimental diets.[4] The rats were individually housed in stainless steel cages. Food and water were supplied *ad libitum*. The diet was a semi-purified type, manufactured in our own kitchens, and complete except for iron (Table 1)[4] (13). Initially, the rats were fed the iron-deficient diet (4.0 mg iron/kg diet). On determination that the rats were clinically anemic, they were divided into 6 groups. Each group was fed the same diet containing varying levels of iron added as ferrous sulfate for 3 weeks (0, 5, 10, 15, and 20 mg iron/kg diet)[4].

In the sixth week of the experiment 2 animals died unexpectedly on successive days. The first animal was from the group receiving 20 mg added iron/kg diet. The second animal was from the group fed the iron-deficient basal diet with no added iron. Necropsies were performed.

Both animals were found lying on their right side. The abdomen in both cases was distended with gas. Gross examination of the abdominal contents revealed that the cecum was enlarged, distended with gas, and hemorrhagic. The colon was empty of ingesta but contained gas bubbles. The stomach, duo-

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[2] This paper reports research undertaken at the US Army Natick (Mass) Laboratories and has been assigned # TP-1071 in the series of papers approved for publication. The findings in this report are not to be construed as an official Department of the Army position.
[3] Accepted for publication December 23, 1971.
[4] Thomas MH: US Army Natick Laboratories, Natick, Massachusetts, Unpublished data.

TABLE 1

*Basal diet for iron deficiency study*

| Component | Amount (grams/100 grams diet) |
|---|---|
| Dried skim milk, non fat | 40.00 |
| Degermed yellow cornmeal | 25.00 |
| Glucose, monohydrate | 23.75 |
| Gelatin, plain, unflavored | 5.00 |
| Corn oil | 3.00 |
| Monosodium phosphate | 1.00 |
| Calcium carbonate | 1.00 |
| Iodized salt | 0.50 |
| Trace mineral premix | 0.45 |
| Choline chloride | 0.15 |
| Vitamin pre-mix | 0.10 |
| dl-methionine | 0.05 |
| Ferrous sulfate | QS for study |

denum, jejunum, and ileum appeared normal, as did the rest of the abdominal organs. Close inspection of the cecum revealed that it had rotated approximately 630° counter-clockwise when viewed ventrodorsally. Further, because in the rat the mesenteric attachment of the cecum is adherent to the terminal ileum, the ileum was carried around the colon the same 1¾ turns, effectively closing the ileo-cecal-colic junction. Heart, lungs, brain, and other organ systems appeared normal. Histopathologic examination of the cecum revealed only postmortem autolysis. At the termination of the experiment all animals were necropsied. While other disease processes were observed (*eg*, chronic respiratory disease), no other animal had gross pathology of the gastro-intestinal tract.

## Discussion

Because the authors could not define a mechanism for the spontaneous occurrence of cecal rotation and acute death, several dietary and surgical manipulations were performed in an attempt to reproduce the condition. Dietary iron levels were ruled out as an influencing factor since both extremes (high and low) of the test groups were involved. There had been problems with diarrhea throughout the study which were attributed to high (25%) levels of lactose (main carbohydrate source). Of the 2 animals, 1 (low iron diet) had diarrhea for 1 week prior to death. The other rat (high iron diet) never had diarrhea, although 4 others in this grouping had diarrhea for varying lengths of time. Six rats from a surplus-animal group were changed from the normal diet (Purina Laboratory Chow[5]) to a balanced diet containing a high (25%) level of lactose as the main carbohydrate source. Within 12 hr, all 6 rats had a watery diarrhea. However, after 10 days on this diet all 6 were alive and thriving despite the continued diarrhea.

Another group of 6 rats was anesthetized and prepared for surgery. In this group the cecum was isolated and manually rotated counter-clockwise for 1, 2, and 3 complete turns (2 rats each). All 6 rats recovered from the surgery uneventfully. At the end of 4 days these rats were killed and necropsied. Examination revealed that the cecal rotations had, in each case, counter-rotated to a normal position, and no gross pathology was produced.

In another group, 6 rats were placed on the high-lactose diet and the diarrhea was produced. Then the cecum was rotated as before. This time, in three rats, a suture was applied to secure the ileo-cecal junction to the right ventro-lateral aspect of the abdominal wall, and in 3 additional rats, a ligature was applied around the ileo-cecal-colic junction itself. One author has stated that death occurs in 6–10 hr after cecal torsion occurs (**12**). Other authors have indicated that 1 to several days were necessary (**6, 8**). The rats prepared in the above described manner all died, but at varying times. One rat in each group died on succeeding days, *ie*, 1 each at approximately 24, 48, and 72 hr. At necropsy each had severe peritonitis which was not seen in the original 2 animals. Also, in the original 2 animals the twisted segments of intestine involved a very narrow area at the ileo-cecal-colic junction with very little involvement of the associated mesentery. In the experimental animals the involved

[5] Ralston Purina Co, St. Louis, Missouri.

segments of intestine covered a larger area and involved more mesentery. This raises the question of the influence of adhesion formation in the pathogenesis of death from cecal torsion. It is reasonable to suggest that adhesions prevent counter rotation.

Cohrs stated that death occurs as a result of a combination of various causes: 1) putrefactive autointoxication, 2) acute anemia from extreme congestion and transudation, 3) tympany with displacement of the diaphragm forward and compression of the lungs and heart and, 4) rupture of the bowel with subsequent absorption of toxic products (14).

Other authors (5–11) have shown the effects of diet and microflora on the size of the cecum in the gnotobiotic rat. However, in the present work the effect of diet was discounted because of the isolated incidences in the study group; *ie*, 2 animals out of 100 receiving basically the same diet. Table 1 has been included to emphasize this point. The microflora of the affected ceca in the 2 animals necropsied from the iron study were not investigated because the materials to do so were not available.

Why the ceca became enlarged initially is unknown. However, we suggest tympany and putrefaction with subsequent toxemia as the pathogenesis. The inciting etiologic factors producing the original enlargement and torsion of the ceca are unknown. However, tympany and putrefaction with subsequent toxemia may be the pathogenesis in the cases reported herein.

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